DIP #605

The Extremely HY RBC Podcast (Step 1-3)

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- **Dr Mohammed Zaki**
https://chat.whatsapp.com/L6dShASc3f97cwHZgCD6Xo

Welcome. My name is Divine. This is episode 605 of the **Divine Intervention Podcasts**. I'm going to be addressing a topic that is ridiculously **high yield** to know for your exams. In fact, I'm going to call this the clutch **red blood cell** podcast.

- The stuff that I'm going to discuss here, you're going to see tested so many different ways on your exams.
- Again, our friends at the NBME means they have a pretty strong emphasis on **hematology**.
- So I really want to make sure that you have a solid basis.
- You may notice that quite a number of my podcasts in recent times have been tilted towards some kind of bizarre topics.

Red Blood Cell Physiology & Basics

- **Red blood cells** are one of those bizarre, bizarre topics, so I think that's a very important thing to kind of keep at the back of your mind, right?
- Let's go ahead and talk about **red blood cells**.

- We know that **red blood cells**, they're also known as the **erythrocytes**.
- And I'm going to make so many integrations, as we go along, right?
- But then as the **erythrocytes** and we know that their job is pretty straightforward, right?
- Well, it involves a lot of processes, but generally pretty straightforward.
- They essentially carry oxygen from your lungs to your peripheral tissues, that's literally their job.

- And then don't forget that there is this thing called **reticulocytes**, right?
- These are like immature **red blood cells**, right?
- The **red blood cells** are immature, and you may wonder why would I be making **red blood cells** that are immature? That doesn't make any sense.
- Well, the reason you do that is if you're in a situation where you're kind of running out of **red blood cells** fast, right?
- You're like, hmm, interesting. I need to figure out a way to deal with this **red blood cell** issue, right?
- So you're like, hey, even if they're immature, doesn't matter, I'm just going to pump them out.

- So typically you're going to see that when you have a condition of **hemolysis**, right?
- So your **red blood cells** are not living for 120 days like they should.
- So you keep just pumping out, pumping out **red cells**, even if they're immature **red blood cells**, right?
- Even if you've lost—and that also happens when you've lost a ton of blood very quickly—your body's like, "Oh no, I need to replenish my **red blood cell** count," right?
- And then you just pump out, pump out, pump out as many as you can **Reticulocyte**, right?
- So whenever you have a higher **retic count**, it tells you that your **bone marrow** is probably doing pretty well.
- It's probably doing pretty well. You're just losing **red blood cells** really, really fast.
- But if your **retic count** is low, it kind of tells you that, umm, maybe your **bone marrow** is not working very well.

- And then we know that **red blood cells**, right, they have a membrane, right?
- We know that that membrane is made up of very important things—made up of a lot of **lipids** and all those fun things.
- I'm going to talk about that in a bit here, right?

- And then they contain **hemoglobin**, right?
- **Hemoglobin** is that actual protein that carries **oxygen**, right?
- It literally has 4 spots on it for **oxygen**, and that's what we call **SaO_2**.
- How saturated is your **hemoglobin** with **oxygen**?
- Well, the thing is, those 4 spots can be taken up by stuff.
- The big one you probably need to know for your exams is **carbon monoxide**, right?
- When **carbon monoxide** binds to that **hemoglobin**, it kind of takes up some space on that **hemoglobin**, and **oxygen** is not able to bind.
- **Carbon monoxide poisoning** is going to crush your **SaO_2**.
- It's going to reduce it.

- Another thing is that **hemoglobin** has **iron** on it.
- It's the **iron** that is the **oxygen** binder, right?
- And **iron** can exist with many charges, but the ones we care about in the body are the **2+ form** and the **3+ form**, right?
- The **2+ form**, that's the **ferrous** form.
- The **3+ form** is the **ferric** form.

- I'm going to tell you something very interesting.
- Out of all the **2+** and the **3+** forms, the body loves the **2+** form.
- The body is like, "Uhm, for the **2+** form, why?"
- Because number one, that **ferrous** form of **iron** is the one that can be reabsorbed in the **GI tract**.
- That's why sometimes when you're taking oral **iron** pills, they say, "Hey, take it with **vitamin C**, take it with **orange juice**," because **orange juice** is a **reducing agent**, so it's going to keep that **iron** in the **2+** form, which increases the **bioavailability** of the oral **iron** supplementation you're taking, right?

- The **Fe^{3+}** form, the **ferric**, cannot be reabsorbed very well.
- And then another thing is it's the **Fe^{2+}** form that can bind **oxygen**, right?

- Whenever you have **hemoglobin** that has **iron** in the **3+** form, what do we call that?
- We literally call that **methemoglobin**, right?
- That kind of **hemoglobin** can literally not bind **oxygen**, right?
- So when a person has **methemoglobinemia**, you have the **iron** in **hemoglobin** in a form that cannot carry **oxygen**.
- Because it cannot carry **oxygen**, guess what?
- Your **SaO_2** is also going to plummet in a person that has **methemoglobinemia**.
- Remember, we're going to give **methylene blue** for that.
- **Methylene blue** catalyzes the conversion of **Fe^{3+}** to **Fe^{2+}**.
- And that's going to be very, very helpful.

- Alright, so that's kind of like the brief introduction I kind of wanted to give you with your **red blood cells**.

- And many of you have heard of the term **hematocrit**.
- Remember, your **hematocrit** is pretty much triple your **hemoglobin**.
- That's a very good approximation to make.
- So if your **hemoglobin** is like 7, your **hematocrit** is triple that—that's 21, right?

- And then many of us are familiar with things like **MCV**, right?
- That's the size of the **red cell**.
- You should be roughly between 80 and 100 femtoliters, right?

- And then we know about the **MCHC**—mean corpuscular **hemoglobin** concentration.
- Hey, how concentrated is the **hemoglobin** within the **red cell**, right?
- So remember, concentration is mass over volume, right?
- So if you mess up the volume of the **red blood cell** in any way, shape, or form—let's say you reduce it because you have **spectrin**, **ankyrin**, or **band protein mutations**—you don't have any **hemoglobin** problem, but the container is just not enough.
- So the **hemoglobin** is going to get very concentrated.
- So your **MCHC** is going to rise.

- So I think let's go ahead and just make a few integrations with **red cells**.
- And I feel like one area that's kind of useful to make integrations with are the **shapes of red cells**.
- There are many ways that they like to kind of go after this on exams, right?

- So we know that the regular shape of a **red cell** should be the **biconcave disc** shape, right?
- But sometimes you can begin to see some **abnormal shapes**, and I will encourage you, as you're listening to this podcast, just pull up your computer and just kind of look up these shapes that I'm talking about, right?

Shapes

Spherocytes

- But the first shape I want to talk about is the one many of us are familiar with: **spherocytes**, right?
- Whenever you see a **spherocyte**, right, you're pretty much just going to see all red.
- You're not going to see any light area within the **red blood cell**, right?
- Typically, whenever you see **spherocytes**, always think of a **membrane defect**.

- Think of something—either the membrane is defective, the membrane of the **red cell** is defective, or there is something that is just slamming hard at that **red blood cell membrane** and destroying it, right?

- So like, for example, **hereditary spherocytosis**—it's a **membrane defect**, right?
- **Autosomal dominant**.
- These we tend to find in Europeans, right—especially Northern Europeans.
- And you notice that these people don't have **spectrin**, they don't have **ankyrin**, they don't have these **band proteins**.
- So the **red blood cell membrane** is not great, right?
- So they will go ahead and assume the **spherocyte** shape.

- Another thing that can cause **spherocytosis** is **autoimmune hemolytic anemia**, right?
- And you may be like, "Divine, why would **autoimmune hemolytic anemia** cause a **spherocyte**?"
- Well, that should make sense—you're literally making autoantibodies against the **red blood cell membrane**, for example.
- So if you are attacking that **red blood cell membrane**, you're destroying it, right?
- You're losing membrane, so you're going to assume that **spherocyte** shape.

- So you may be like, "Oh fine. How am I going to differentiate **hereditary spherocytosis** from **autoimmune hemolytic anemia**?"
- It's actually pretty straightforward—do the **Coombs test**, right?
- Because remember, in **hereditary spherocytosis**, it's not **autoantibodies** that lead to destruction of the **red cells**.
- But in **autoimmune hemolytic anemia**, that's a **type II hypersensitivity reaction**.
- It's **autoantibodies** that lead to that happening, right?
- So honestly, just use the **Coombs test**.
- The **Coombs test** will be **negative** in **hereditary spherocytosis**, but will be **positive** in **autoimmune hemolytic anemia**.

Rouleaux formation

- Alright, next shape I'm going to talk about is the **Rouleaux formation**, right?
- Many of us are pretty familiar with the **Rouleaux formation**.
- And I know when I say **Rouleaux formation**, the first thing many people say is, "Oh Divine, that is **multiple myeloma**!"

- Well, what's the mechanism behind the **Rouleaux formation**?
- See, **red blood cells**—let me tell you something cool about them.
- Their membranes are **negatively charged**.
- So **red blood cells** don't like to hang out with each other, right?
- Because remember, **like charges repel each other**, right?
- So **red cells** don't like to hang out with each other because their membranes **repel** each other.
- But if, for whatever reason, you have a lot of **proteins** in the bloodstream that have a **positive charge**, all those **proteins**, as they float around, diminish the strength of the **negative charge** generated.
- They literally diminish the strength of the **negative charge** generator.
- So that makes **red cells** say, "Oh hey dude, you don't have as much **negative charge** as I'm used to seeing—come on, let's get married!"
- And the **red blood cells** start **stacking up** with each other like **coins**.
- So whenever you have like four or more **red cells** stacked like coins, we call that the **Rouleaux formation**.

- If you understand that mechanism, then the causes I'm going to describe will make a lot more sense to you.
- For example, if you have **multiple myeloma**, you have all these **monoclonal proteins** accumulating—those things have **positive charges**.
- They will diminish the **negative charge** on the **red blood cell membranes**, and the **red cells** are going to start stacking.
- You'll also see this in things like **Waldenström's macroglobulinemia**, for example.

- In fact, let me shock you—when you have a lot of **inflammation** going on in the body, you're also going to have **Rouleaux formation**.
- Why? Because when you have **inflammation**, you're making a ton of **antibodies** to help deal with whatever bug is causing problems.
- That's going to cause **Rouleaux formation**.

- In fact, let me ask you this: What do you think is the **most common cause** of **Rouleaux formation**?
- I hope you're not saying **multiple myeloma**.
- I hope you're saying **pregnancy**.

- **Pregnancy** is the **most common cause** of **Rouleaux formation**.
- Why? Because in **pregnancy**, you make a ton of **proteins** from the liver, like **fibrinogen**, for example.
- **Fibrinogen** has a **positive charge**.
- It diminishes the **negative charge** on the surface of **red blood cells**, increasing interactions between them and causing **Rouleaux formation**.
- And I guess this is kind of a side point: this is partly why you have a lot of **hypercoagulability** in **pregnancy**, right?
- Because you're making a lot of **fibrinogen**.
- Remember, **fibrinogen** is that thing that binds to **GP IIb/IIIa** from your **platelets** to make a **platelet plug**, right?
- That's one.

- But two, you also increase the production of **clotting factors** during **pregnancy**, right?
- So that means **secondary hemostasis** is running really good, right?
- That **coagulation cascade** can make you get in trouble.

- And you may wonder, "Divine, why is it that the body goes through the pains of making more **fibrinogen** and of making more **clotting factors** during **pregnancy**?"
- It's actually for a pretty simple reason.
- Your body knows that it's about to go through a pretty bloody process, right?
- Again, I don't know about you, but I remember during my **OBGYN** rotation—even for a pretty normal delivery—a lot of **blood** is kind of coming out from this woman's **vagina**.
- The body wants to be prepared and wants to make that **bleeding** stop.
- So you want to beef up your **platelet** cascade.
- You want to beef up your **clotting factor** cascade, right?
- So that kind of makes sense.

- And many times, the synthesis of many of these things is driven by **estrogen**.
- You literally make tons of **estrogen** during **pregnancy**.
- Now, I think that's enough integrations with the **Rouleaux formation**—kind of cool stuff there, right?

Elliptocytes

- Next one I want to talk about: let's talk about **elliptocytes**, right?
- **Elliptocytosis**—another name you may see for **elliptocytosis** is **ovalocytes**.

- You're going to see this with something like **hereditary elliptocytosis**—that's kind of the big thing to know there.
- Basically, the **red blood cell** will look longer than it is wide.
- That's **elliptocytosis**.
- That's about as much as I'm going to say about that one.

Dacrocyte / Tear Drop Cell

- Another **red cell** kind I want to talk about is the **dacrocyte**, right?
- The **dacrocyte**—sometimes we call this a **teardrop cell**, a **teardrop cell**.
- So you see a **red blood cell** that kind of looks like a **teardrop**, right?
- Like the kind of tears people shed after they take their **USMLE exams** or a tough **UWorld** block, right?

- The thing that happens is when you see **red cells** that have that shape, it means those **red cells** really struggled to get out of the **bone marrow**.
- So why would **red blood cells** struggle to get out of the **bone marrow**?
- They're going to struggle to get out if the **bone marrow** contains a ton of **collagen**, right?

- So let's say there's a lot of **fibrosis** in the **bone marrow**.
- What will cause you to have **fibrosis** in the **bone marrow**?
- The big, big, big one you want to know for your exams is **primary myelofibrosis**.
- You're making all these things from **megakaryocytes**, like I believe **platelet-derived growth factor** or whatever.
- That's going to stimulate **fibroblasts** to lay down a ton of **collagen**.
- As your **red cells** are squeezing through all that **collagen** to get out of the **bone marrow**, they're going to assume that **teardrop shape**.
- We call those **dacrocytes**, right?

Acanthocyte / Spur Cell

- Another classic **red cell** shape you may want to be familiar with on your exams is the **acanthocyte**, right?
- Another name for an **acanthocyte**, by the way, is a **spur cell**.
- Again, please make sure you know these alternate names.
- Our friends at the **USMLE** love using derivatives.
- They will literally take what you know and just put it in different terms.
- So make sure you can identify that, hey, an **acanthocyte** is also a **spur cell**.

- So what causes people to have an **acanthocyte**?
- Well, the big one you should know for your exams is when you are having issues with your **red blood cell membrane lipids or proteins**, right?

- Basically, an **acanthocyte** looks like a **red blood cell** that has **thorns** coming off of it.
- So what are some things that can cause you to have **lipid or protein** issues in your **red cell**?
- Easy—big one is this disorder known as **abetalipoproteinemia**.
- **Abetalipoproteinemia**, right?
- What causes **abetalipoproteinemia**?

- It arises from an **MTP mutation**—a **microsomal triglyceride transfer protein mutation**, right?
- **MTP** helps you assemble **apolipoproteins** in your **enterocytes** of your **GI tract**.
- So **apolipoproteins** are things like **chylomicrons** and all those fun things.
- Those **chylomicrons** and all those **apoBs** or whatever—they help you move **lipids** into your **circulation**, right?
- So if those things are not assembled well, you won't be able to move **lipids** and **lipid-soluble vitamins** like vitamins **A, D, E, and K**—especially **vitamin E**.
- At least, that's the one we're concerned about for these kinds of cells—**vitamin E** won't be able to move in well.
- And you need **vitamin E** for **membrane production**, right?
- So you're going to struggle with that, and you're going to have **acanthocytes**.

- Obviously, if you have **liver disease**, right—if your **liver** is not working very well—you won't be able to make **lipids** very well.
- Your **liver** is a very big **lipid** producer, right?
- So if your **liver** is not working very well, for example, you won't make **proteins** very well either.

- Think about it: why do you think people that have **liver disease** have **ascites**, right?
- And they have **edema**?
- Because they're not making **albumin**.
- **Albumin** is a very critical **protein** in the body.
- So your **oncotic pressure** will plummet.
- That's how you get that **edema**, right?

- But again, if you have **liver disease**, you are not making **lipids** well, you are not making **proteins** well—so you're going to have these **acanthocytes** popping up a lot.
- Just something to keep at the back of your mind.

- Also, if you're just not eating enough **lipid** or **protein**, right?
- So believe it or not, a person who's **anorexic**—especially in severe **anorexia**—you're going to see a bunch of **acanthocytes** on their **blood smear**.

Sickle Cells

- And then, obviously, **sickle cells**—you're going to see this when you have **sickle cell disease**, right?
- You may wonder, what's the mechanism behind the formation of a **sickle cell**?
- Why does the **red cell** assume that shape?
- It's because you have a lot of **hemoglobin S**.
- **Hemoglobin S** is very, very good at **polymerizing**, kind of partnering up, right?
- A bunch of **hemoglobin S** molecules—when they **polymerize**, they form those abnormal shapes.
- So it's due to **polymerization of hemoglobin S**—that's how you get **sickle cells**.
- You can see that in **sickle cell disease**, right?

Schistocytes / Helmet Cells / Fragment Cells

- And then **schistocytes**—**schistocytes** are another very classic one.
- And again, there are many names they use for **schistocytes** on the **USMLE**.
- They can call them **helmet cells**, they can call them **fragment cells**.

- But what is the thing that—what is the **mechanism**?
- Again, I want you to **understand** things and not just memorize them.
- That's one of the purposes of this podcast.
- It's not just to prepare for exams, but it's also to help you understand medicine well.

- What's going to cause a **schistocyte**?
- Well, a **schistocyte** is going to be caused when, for whatever reason, you have something that **disrupts the red cell membrane**—think of it like a **nail** ripping apart the **red cell membrane**.

- You have a **traumatic impingement** on the **red blood cell membrane**.
- So what are those things that can cause that **traumatic impingement**?
- Very big one is what we call **MAHA**.
- Many of you have heard of the term **MAHA**.
- What in the world does **MAHA** stand for?
- Well, **MAHA** means **microangiopathic hemolytic anemia**.

- Let me ask you this—is there another name they use for **MAHA** on your exams?
- Yeah, they may call it **thrombotic microangiopathy**.

- Basically, you have a bunch of these **platelet thrombi** just kind of hanging out in your **circulation**, in your **bloodstream**.
- Those things are like **big sharp sticks** hanging out in your bloodstream.
- They can rip your **red blood cell membrane** apart.

- So what are some **causes of MAHA**?

- Don't forget things like **HUS**—**hemolytic uremic syndrome**.
- Don't forget things like **TTP**—remember in **TTP**, you have an **ADAMTS13 deficiency**.
- **ADAMTS13** is needed to break down or dissolve **von Willebrand factor**.
- But if you have a **deficiency** of that enzyme, you won't break down **vWF**, so you'll be making all these **platelet thrombi**.
- You'll have more of those **very sharp pointy sticks** in your bloodstream.

- **DIC**—**disseminated intravascular coagulation**—is another cause of **MAHA**, right?
- **HELLP syndrome**—**hemolysis, elevated liver enzymes, low platelets**—that's another cause of **MAHA**.
- **Preeclampsia**, **eclampsia**—those are also causes of **MAHA**, right?

- Also, people that have really **high blood pressures**, like **hypertensive emergencies** or whatever, that can also certainly cause a person to have **schistocytes** on a **blood smear**, right?

- Or if your **vessel walls** are inflamed, like in **vasculitis**, that can also cause **schistocytes** on a **blood smear**, right?

- Or if your **kidney vessels**—like your **glomeruli**, for example—have a lot of **inflammation**, like you have **glomerulonephritis**, that can also cause **schistocytes** on your exams, right?

- Or guess what—they can even give you a question about a person that has **jaundice**, and you just placed a **prosthetic valve** for them because they had some kind of **valvular disorder**.
- That can also cause **schistocytes**, right?
- **Prosthetic valves** are very good at **shearing red blood cells** under certain circumstances.
- As they shear those **red cells**, you're going to make a lot of **indirect bilirubin**.
- That's going to cause a lot of **jaundice**, right?

Target Cells / Codocytes

- So many of you have also probably heard of **target cells**, right?
- **Target cells** are sometimes called **codocytes** on the exams.
- It's almost like having a **bull's-eye red blood cell**, right?
- Think of this with **thalassemia**.
- Think of this with **liver disease**, right?
- We don't need to worry about the mechanism behind that—that's a little too much for your tests.

- So again, I would know these different integrations that I've made.
- I think it's going to be very helpful to you on your exams, right?

Inclusion Bodies

- And also, don't forget some of these **inclusions**—these things you can find within **red blood cells**, right?
- For example, remember: a **red blood cell** should **not** have a **nucleus** in it.
- Maybe if you see an immature cell, you may see some **nuclear material**, but a mature **red blood cell** should not have a **nucleus**.

Heinz Bodies

- Then don't forget **Heinz bodies**.
- What are **Heinz bodies**?
- Make sure you know what **Heinz bodies** are associated with—obviously **G6PD deficiency**.
- These are basically **hemoglobin** molecules within the **red cell** that have been **denatured** and **precipitated**.
- We're going to see this when there's a lot of **oxidative stress** in a **red blood cell**.

- For example, if a person has **G6PD deficiency**—remember that's an **X-linked recessive** disease where you **cannot make NADPH**.
- If you can't make NADPH, you can't regenerate **glutathione**.
- Without **glutathione**, you'll have a lot of **oxidative stress**, which causes the **hemoglobin** to **denature and precipitate**, creating a **Heinz body**.

Bite Cell

- Then, when a **red cell** containing those **Heinz bodies** gets to the **spleen**, the **spleen macrophages** will say, "What in the world is this?" and **eat it up**.
- That creates a **bite cell**.
- Many people confuse this with a **Howell-Jolly body**.

Howell-Jolly body

- **Howell-Jolly bodies** are basically **nuclear remnants**.
- Normally, they are removed when the **red cell** reaches the **spleen**.

- The **spleen** is like a amazing a**quality control center** for **red blood cells**.
- If your **spleen** is **absent**, then you can't remove those remnants—so you end up seeing **Howell-Jolly bodies**.
- So you can see this in a person who has sickle cell disease.
- Remember, by age four, their spleens are practically gone, right?
- Or if a person has had a splenectomy for any reason—remember, splenectomy is considered a third-line treatment for ITP (immune thrombocytopenia).
- It's also used as a treatment for certain hereditary spherocytoses, right?
- That's why such people may start developing Howell-Jolly bodies.
- Remember, seeing Howell-Jolly bodies on your exam does not always mean sickle cell disease specifically.
- You can see them in any condition where the spleen is absent or non-functional.

Basophilic Stippling

- Then don't forget that **basophilic stippling**—we tend to see this as basically **ribosome aggregates**.
- **Ribosome aggregates** you see in a **red blood cell**.
- Many times, you're going to see this in a person that has **lead poisoning**, right?

- Just **heavy metal poisoning** in general, but **lead poisoning** is probably the big one to know for your exams.
- That typically is going to be associated with **basophilic stippling**.
- Again, these are **aggregations of ribosomes**.
- They're going to be **blue**.
- Don't forget that color—they're going to be **blue**.
- So they're stippled—**blue dots** within the **red cell**.

Sideroblasts

- Many of us know that **basophilic stippling** has a tight association with **sideroblasts**, right?
- What are **sideroblasts**?
- Basically, **sideroblasts** are **collections of iron** in the **cytoplasm**.

- And why would that **iron** collect?
- Well again, the big cause you need to know for your exams is **lead poisoning**.
- When you have **lead poisoning**, remember, **lead inhibits many enzymes** in **heme synthesis**, like **ferrochelatase** and **ALA dehydratase**.
- So you cannot make **protoporphyrin**.

- And you may wonder, what's the point of **protoporphyrin**?
- The thing is, **protoporphyrin binds to iron to make heme**.
- So if you can't make **protoporphyrin** because you've inhibited the enzymes that produce it, then **iron** will just be sitting there waiting—
- Like, "Come on bro, I've been waiting to meet you!"
- That **iron** is going to gang up in the **mitochondria** of the **red blood cell** and create that **ring sideroblast** shape.
- That's actually something that's pretty **high yield** to know for your exams, right?

- Or say, for example, you have a **vitamin B6 deficiency**—because remember, you need **B6** to make **heme**, right?
- If you remember the first step of **heme synthesis**, I think it's **glycine** and **succinyl-CoA**, they kind of come together.
- I'm pretty sure the enzyme that does that is **ALA synthase**.
- That uses **B6** as a **cofactor**.

- So if you get a **B6 deficiency**, that can cause you to run into problems, right?
- So just kind of keep that at the back of your mind.

- **Ring sideroblasts**—you can see that in **B6 deficiency** (taking isoniazid), you can see that with **lead poisoning**, and you can also see that in people who consume a lot of **alcohol**.
- And one important cause of **sideroblastic anemia** as well is **myelodysplastic syndrome**.

- **Myelodysplastic syndrome** is a pretty **high yield** cause of **sideroblastic anemia**.
- The mechanism behind that—you don't need to worry about it for your exam, so we're going to skip that.

Banana Shaped Inclusions

- And then the last **inclusions** I'm going to talk about—remember those **banana-shaped inclusions** that you find within **red cells** in people who have **malaria**, right?
- So like infection with **Plasmodium species**.

Maltese Cross Pattern

- And then don't forget that **Maltese cross** pattern you'll find within **red cells** when a person has **babesiosis**, right?
- That's going to be a person who visited **New England** and got bitten by an **Ixodes tick** causing problems for them, right?
- Because remember, the **Ixodes tick** also carries **Babesia** in addition to **Anaplasma** and **Borrelia burgdorferi**.
- So I think this is kind of a good stopping point.
- But again, this podcast is extremely **high yield**.
- You're going to see this stuff tested very, very heavily on your exams.